DISSERTATION ON

DIABETIC FOOT ULCERS

M.S. DEGREE EXAMINATION

BRANCH-I

GENERAL SURGERY

THANJAVUR MEDICAL COLLEGE

THE TAMILNADU DR.MGR MEDICAL UNIVERSITY

CHENNAI

SEPTEMBER 2006

CERTIFICATE

This is to certify that this dissertation entitled **"DIABETIC FOOT ULCERS"** is the bonafide work done by **Dr. R.R. RAMKUMAR** submitted as partial fulfillment for the requirements of **M.S. Degree Examinations Branch I, General Surgery, SEPTEMBER 2006.**

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ACKNOWLEDGEMENT

It is with profound gratitude that I thank my beloved chief and guide **Prof. Dr.G.AMBUJAM, M.S., FICS.,** for her guidance and constant encouragement, without which, this work would not have been possible.

It is with the utmost respect and thankfulness that I place on record, the role of the former HOD and my former chief **Prof. Dr.R.THIRUNAVUKKARASU, M.S., DLO.,** during the initial period of this work.

I consider it a privilege to have done this study under the supervision of the Professor & Head of the Department **Prof Dr. V.THIRUGNANAM, M.S., M.Ch.,** who was a constant source of inspiration, whose suggestions were invaluable in refining this study.

I am extremely thankful to **THE DEAN**, Thanjavur Medical College, for granting me permission to undertake and complete the dissertation in the Thanjavur Medical College Hospital, Thanjavur.

I am also grateful to **Prof. Dr.A.Devakumari, M.S.,** Professor of Operative Surgery, **Prof.Dr.R.M.Natarajan, M.S.,** Professor of Principles and Practice Of Surgery, **Prof.G.Venkatesan, M.S.,** Professor of Surgery and

Prof.Dr.R.Marudavanan, M.S., Professor of Surgery for their role in shaping this study.

My grateful thanks to **Dr.T.Anantharamakrishnan, M.S., Dr.T.Krishnamoorthy, M.S., Dr.S.Maruthu Thurai, M.S.,** and other Asst. Professors for their valuable opinions and moral support while doing this dissertation.

I am also grateful to the Professors and Assistant Professors of Radiology Department and Pathology Department for untiringly assisting me during this study.

Most of all I owe my sincere thanks to all the patients who have participated in this study with remarkable endurance and co-operation to whom I intend to dedicate this study.

CONTENTS

Introduction

- **For the past few decades there has been an alarming rise in the prevalence of diabetes particularly type II DM.**
- **This correlates with the rising rates of obesity.**
- **In the diabetic population, foot complications are a major cause of hospitalization.**
- **20% of diabetics develop some foot problems during the course of their illness.**
- **The foot ulcers are characterized by an inability to self-repair in a timely and orderly manner.**
- **Risk factors for Diabetic foot ulcer are: -**
    i. **Long Diabetes duration.**
    ii. **Peripheral neuropathy**
    iii. **Peripheral vascular disease**
    iv. **Prior foot ulcers**
    v. **Prior amputations**
- **The prevalence of neuropathy in diabetics is 23%.**
- **The prevalence of peripheral vascular disease is 15%.**
- **About 50% of the diabetic foot ulcers require a minor or major lower limb amputation.**
- **About 45% of all lower limb amputations are performed for Diabetic foot disease.**
- **Foot ulcers result in amputations in about 50% of patients.**
- **Once one limb has been amputated, the patient's 5-year survival rate is only about 30%.**
- **Thus the morbidity extracts a considerable toll not only on health care provision but especially on patients.**
- **We should strongly endorse a national initiative, which should parallel the advances and understanding of the pathophysiology and treatment so as to create diabetic foot clinics to combat this challenge.**

HISTORICAL REVIEW

The term DIABETES meaning “to pass through” was first coined by Aretaeus in 2nd century AD.

The sweet taste of diabetic urine was noted in 6th century AD by an Indian physician Susrutha who called it as “medhu meha”

The term “DIABETES MELLITUS” was coined by John Rollo in 18th century. Claude Bernard described the metabolism of diabetes in 19th century.

Insulin was discovered at the university of Toronto in 1921, by Frederick G Banting a surgeon, and Charles H. Best, a student assistant.

The American physician Elliot P. Joslin was one of the first doctors to gain experience with insulin.

The Cambridge scientist Frederick Sanger described the primary structure of insulin in 1955 for which he was awarded the Nobel Prize in 1958.

Amputations as a form of therapy date back to Hippocrates (460-377 BC)

Ambrose Pare (1510-1590) is considered to be the originator of modern principles of amputation surgery.

During the Napoleonic wars, Dominique Jean Larry (1776-1842) continued and expanded the principles of amputations.

AIM OF THE STUDY

1. To study the percentage of patients undergoing amputation for diabetic ulcer of foot.

2. To study the percentage of biopsy proved neuropathic changes in diabetic foot ulcer.

3. To study the common organisms causing foot infection and the sensitivity of the drug in patients with diabetic foot ulcer.

4. To study the percentage of vascular changes in diabetic foot ulcer.

5. To study the number of patients showing osteomyelitis in Roentgenogram.

MATERIALS AND METHODS

The clinical material for this study consisted of 50 cases of Diabetic foot ulcer patients admitted in the surgical wards of Thanjavur Medical College Hospital, Thanjavur during the period 2003 to 2006.

The patients were selected based on the following criteria.

1. Presence of Diabetes mellitus
2. Patients with major trauma as a cause of foot ulcer are excluded.
2. Thermal burns or scalds are excluded.
3. Patients with Hansen's disease leading to trophic ulcer are excluded.
4. Patients with filariasis and its sequelae are excluded.

The following data were collected and recorded.

In the proforma, name and age of the patient, sex, occupation, complaints and history in detail were obtained and recorded. Past history of diabetes, hypertension, tuberculosis and ischemic heart disease were enquired into. Smoking and alcohol history were elicited with special reference.

Patients were examined in detail about the general condition like anemia, jaundice, fever, blood pressure and peripheral pulses.

The affected part of the foot the ulcer was examined in detail for all the features of an ulcer with special reference to motor and sensory changes.

The following investigations were carried out as per the need of the patient.

Urine : Albumin, Sugar, Deposit

Blood : Hemoglobin, Total Count, Differential Count, Platelet Count, ESR

Blood : Sugar and Urea.

Serum : Creatinine, Sodium, Potassium, Chloride

ECG and X-ray Chest were taken

Pus culture and sensitivity: Organism and the appropriate antibiotic were identified.

Color Doppler Study : Arterial study for macrovascular diseases.

Skin Biopsy : For Neuropathic changes.

X-ray of the foot affected : To look for osteomyelitis of the underlying bone.

The mode of treatment given - whether Conservative surgical Debridement or Amputation, complication and hospital stay were recorded in the proforma.

Emphasis on the level and type of amputation performed was recorded.

REVIEW OF LITERATURE

Diabetic foot ulcer represents a formidable clinical problem. Successful treatment requires a thorough understanding of the pathologic process, surgical debridement of devitalized tissue and updating various modalities of treatment. Failure to expeditiously recognize the cause, pathology and associated infectious process may lead to devastating consequences, like amputation and death.

Loudon in 1805, had quoted that “Leg ulcers are generally looked upon as an inferior branch of practice, an unpleasant and inglorious task where much labour must be bestowed and little honour gained”.

In 1997, the American Diabetes Association proposed the following diagnostic criteria for diabetes.

1. **Symptoms and Random plasma glucose ≥ 200mg /dl.**
2. **Fasting plasma glucose ≥ 126mg/dl on two occasions.**
3. **75g OGTT 2 hrs plasma glucose ≥ 200 mg / dl.**

Impaired fasting Glucose (IFG)

110 to 125 mg/dl.

CLASSIFICATION OF DIABETES : (ADA 1997)

1. Type 1 diabetes (Immune mediated)
2. Type 2 diabetes.
3. Other specific types.
4. Gestational diabetes mellitus.

COMPLICATIONS

1. Acute a) diabetic ketoacidosis
   b) Hyper osmolar hyperglycemic, nonketotic syndrome.

2. Chronic a) Microvascular disease
   b) Retinopathy
   c) Nephropathy
   d) Macrovascular disease
   e) Neuropathy

MANAGEMENT

Goals : To achieve a

1. Fasting blood glucose 80-120 mg/ dl.
2. Bed time glucose 100-140 mg/dl
3. Hemoglobin A_{1c} less than 7%.

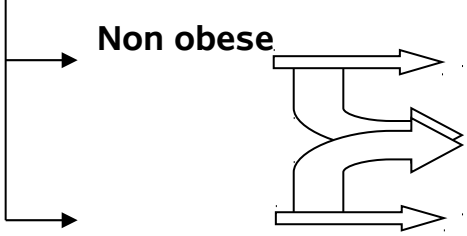
MODALITIES OF TREATMENT :

1. Diet (low fat, low salt, high fibre carbohydrate diet)
2. Exercise
3. Patient education
4. Pharmacological therapy : Sulphonylureas
   Biguanides
   Alpha-glucoside inhibitors
   Thiazolidinediones
   Insulin

TREATMENT PROTOCOL

Less than 40 yrs

Non obese → **Weight maintenance diet**



Insulin

Obese **Low energy diet**

More than 40 yrs

Obese → **Low energy diet, exercise**

± Biguanides or Acarbose

Non obese **Weight maintenance diet**

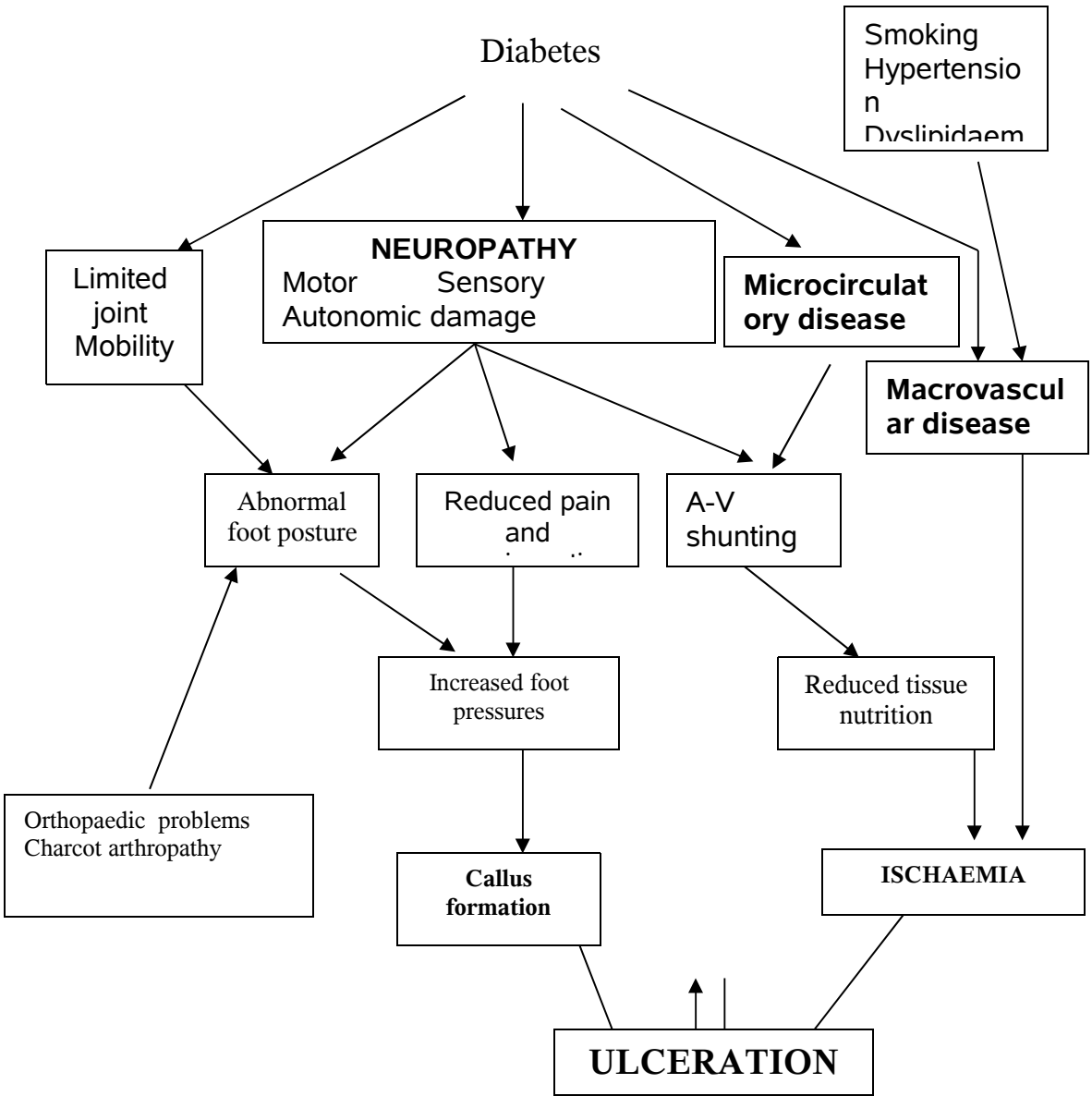
→ ± Sulphonylureas

± Sulphonyl ureas + biguanide

± Insulin

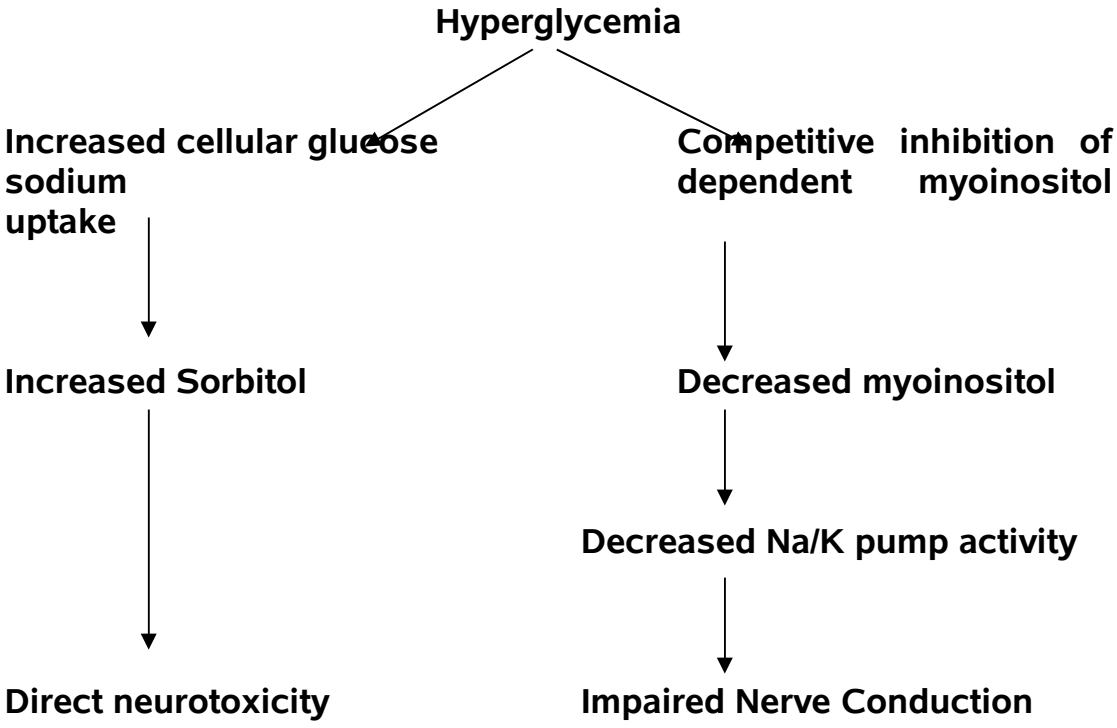
PATHOLOGY OF DIABETIC FOOT ULCER

Multifactorial etiology is characteristic.



1.NEUROPATHY:

Diabetic neuropathy affects the somatic motor, sensory and the autonomic nervous system. Sensory neuropathy can lead to extrinsic neuropathic foot ulceration following trauma. The initial trauma is often minor and may be due to ill-fitting footwear, thermal, foreign bodies in shoes, toenail cutting and thorn pricks. Sensory neuropathy paradoxically produces pain and paraesthesias.



Somatic motor neuropathy results in weakness of the intrinsic muscles of the foot, which allows abnormal movement of the small bones of the foot. As a result, joint subluxation occurs. Attenuation of the Achilles reflex is an early sign of motor neuropathy.

Because of sensory neuropathy, the patient continues to walk on the damaged foot and the bony structure of the foot is altered permanently.

These lead to deformities like claw foot, prominent metatarsal heads, varus malformation and rocker-bottom foot.

Inflammation of these subluxated joints results in Charcot's arthropathy. The bony changes produce localized areas of high pressure on the sole with callus formation, and eventual ulceration.

Automatic neuropathy produces abnormal vasomotor responses. Autosympathectomy increases arteriovenous shunting which leads to osteopenia and functional ischemia. Hypohidrosis results in dry, cracked skin, which increases the susceptibility to ulceration and infection.

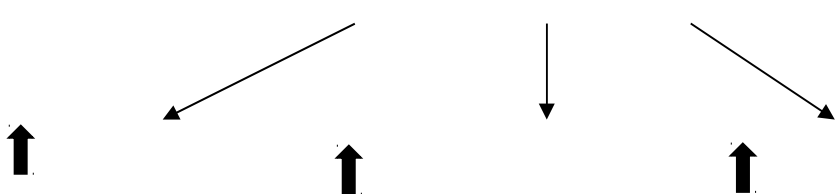
MICROVASCULAR DISEASE :

Due to excessive glycosylation of collagen and glycoproteins, the extracellular matrix is altered. The basement membrane is thickened and there is increased capillary permeability.

Although there is an absolute overperfusion at rest, there remains a relative hypoperfusion of tissues because of increased metabolic rate and lessened ability to vasodilate in response to noxious stimuli.

The vasoconstrictor response to the vertical posture is also reduced with the result that capillary pressure raises. This increases oedema formation with the effect of impairing tissue perfusion.

HYPERGLYCEMIA



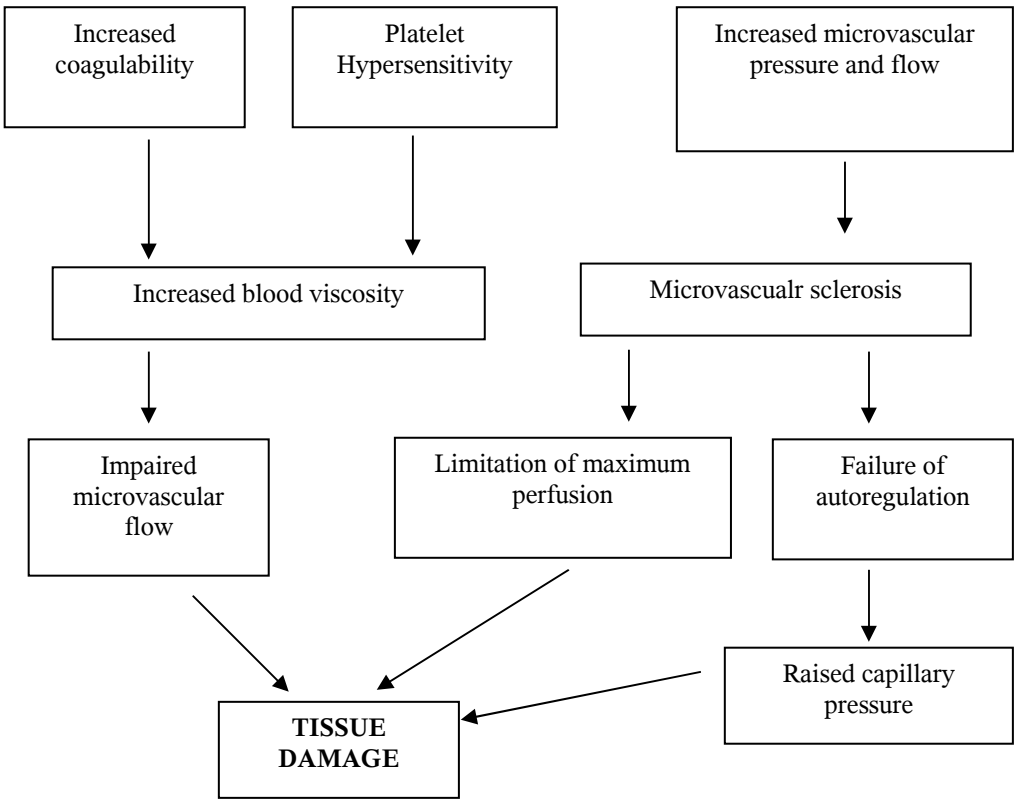
Increased Glucose flux

Increased Diacyl glycerol

Increased Protein kinase C activity

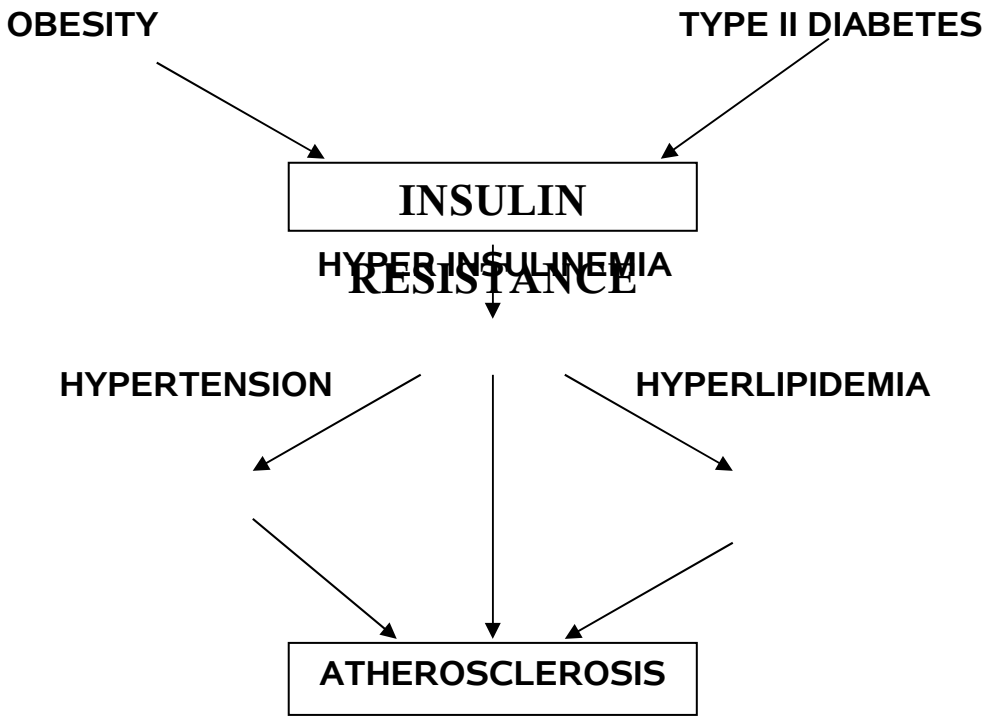
Permeability	Vasoactive hormones Blood flow changes	Basement membrane synthesis

HEMODYNAMIC HYPOTHESIS



Macrovascular Disease :

- Diabetics are 4 to 7 times more prone for atherosclerosis. This process also appears to be accelerated.
- In diabetics, atherosclerosis tends to affect the femoropopliteal and tibial vessels.
- The media of arteries in diabetes tends to become calcified which results in making measurement of ankle brachial indices unreliable.



INFECTION :

- Diabetic foot ulcers are characterized by polymicrobial infection with gram positive and gram negative aerobes and anaerobes.
- Staphylococcus aureus, Bacteroides, Proteus, Enterococcus, Clostridia and Escherichia coli are commonly present.
- Infection is usually secondary.
- Fungal infection between the toes can lead to skin breakdown and secondary ulceration.
- The infection may remain superficial, spreading cellulitis can develop or the infection can spread into the deeper tissues.
- Arterial insufficiency and the diminished inflammatory response facilitates for the rapid progression of any infection.

CONNECTIVE TISSUE ABNORMALITIES:

Advanced glycosylation end products form covalent cross-links with other proteins including collagen and keratin. This cross-linking produces tissues that are rigid, inflexible and resistant to proteases.

The rigidity of the subcutaneous tissue renders it more likely to be torn by shear forces. Wound contraction is also affected.

The collagen of the ligaments and joint capsule become inelastic and weak contributing to foot deformities.

HAEMOTOLOGICAL DISTURBANCE:

Red cells are less deformable possibly due to glycosylation of their cell membrane.

There is a tendency towards hypercoagulability.

White cell function is impaired with the resultant decreased resistance to infection.

MEGGITT WAGNER WOUND CLASSIFICATION

I. **Superficial wound**
II. **Penetrates to tendon or bone**
III. **Deep with osteitis**
IV. **Partial foot gangrene**
V. **Whole foot gangrene.**

DIABETIC FOOT RISK CLASSIFICATION SYSTEM:-

Category 0 Protective sensation intact

Category 1 Loss of protective sensation (LOPS)

Category 2 LOPS + Deformity

Category 3 LOPS + Deformity + H/o previous ulcer or Amputation.

Category 4A Noninfected nonischemic wound

Category 4B Acute Charcots Arthropathy

Category 5 Diabetic foot infection

Category 6 Critical ischemia

INVESTIGATIONS:

For proper management of Diabetic foot ulcers, the most important thing is a thorough clinical examination and a few specific investigations for vasculopathy and neuropathy.

BASELINE TESTS :

- **Urine analysis**
- **Complete blood count**
- **Blood sugar – Fasting and Postprandial :**

  To assess the glycemic status and the dosage of insulin required.
- **Blood urea and serum creatinine**

  In all diabetics, renal parameters should be evaluated since renal failure can coexist with diabetes.
- **Serum Sodium and Potassium**

  Serum electrolytes are evaluated since electrolyte imbalance is common in ketotic and non-ketotic hyperglycemia.
- **Plasma Acetone**

  Presence of acetone in the plasma has to be ruled out since ketoacidosis is an acute life threatening complication of diabetes and may also be caused be uncontrolled sepsis and early recognition can reduce mortality and morbidity.

CULTURE AND SENSITIVITY OF ULCER DISCHARGE

The bacteriology of the organism has to be identified and the appropriate antibiotic tested for effective control of infection and fruitful outcome of the treatment.

XRAY FOOT AP AND OBLIQUE

Plain radiographs can identify soft tissue calcifications, presence of gas in subcutaneous plane from bacterial infections, presence of foreign bodies, fractures, bony deformities and osteomyelitis.

SPECIAL TESTS:

ANKLE BRACHIAL PRESSURE INDEX

It is done for peripheral arterial disease. Ankle blood pressure and brachial blood pressure are measured using a Doppler probe.

$$\text{ABPI} = \frac{\text{Ankle blood pressure}}{\text{Brachial blood pressure}}$$

Normal 1 $\pm$ 0.1

Diabetics may have false high ABPI due to vascular calcification.

SKIN BIOPSY IN DIABETIC ULCER :

The biopsy is obtained from non-glabrous skin, which contains unmyelinated nerves to the epidermis, sweat glands- autonomic sudomotor axons and blood vessel-autonomic vasomotor axons. The quality of nerves in the epidermal and papillary dermis is analyzed, with loss of nerves correlating with the duration of diabetes.

Neuropathies occur in the fifth decade to seventh decade. It is rare to develop neuropathic ulcers within the first 5 years of onset of diabetes but 50% of all diabetes have some sign of neuropathy within 25 years of onset of diabetes. (Reference – Hospital Today- September 1999 Vol IV No.9 and O'Brient et al Study on Peripheral Neuropathy in Diabetes).

The histopathological changes appreciated in the nerve ending by punch biopsy of skin as described by Bolton and associates are the following: -

1. Alteration in myelination

2. Decreased fibre density
3. Axonal degeneration
4. Pericyte degeneration in interstitium
5. Endoneural perivascular basement membrane reduplication.

The above changes can be appreciated by routine eosin and haematoxylin staining. Myelin changes can be made out with Luxol fast blue. Epoxy embedded semi thin sections of osmicated tissues is the most accurate method for determining nerve fibres.
(Reference : Diabetic Neuropathy by Dyck and Thomas 2nd edition page 134).

NERVE STUDIES

The Semmes – Weinstein 10 g monofilament is pressed onto the skin until it buckles. If the patient is unable to feel this, the protective sensation is lost. The vibration perception threshold is measured using the biothesiometer. A threshold of greater than 25v indicates significant neuropathy.

DOPPLER ULTRASOUND

Doppler study of the vascular tree gives essential information regarding the presence of macrovascular disease. This is critical in planning further treatment. The level of amputation, if needed, is also decided by the presence of adequate vascularity.

ARTERIOGRAPHY

Arteriography is indicated only if definitive vascular procedure is planned. It shows the site of occlusion of artery and corkscrew pattern of collaterals in chronic ischemia. It is done by Seldinger's technique.

FOOT PRESSURE MEASUREMENTS

The optical pedobarography is a device that measures dynamic plantar presssures. It allows an accurate measurement of high pressures under small areas of foot. However this system is limited to measurements of bare foot pressures.

Over the past two decades, computer technology has enabled microprocessor like sensors to measure in shoe foot pressures. The EMED system and FSCAN system are widely in use now.

MANAGEMENT OF PAINFUL DIABETIC NEUROPATHY

GENERAL MEASURES :

- Improve glycemic control
- Treat other factors
  - Alcohol excess
  - Vit B12 deficiency
  - Uremia
- Patient education and reassurance

Burning pain:

1. Tricyclic antidepressants
   Imipramine
   Amitriptyline ± fluphenazine
2. Capsaicin cream

Lancinating Pain

1. Anticonvulsants
   Carbamazepine,
   Phenytoin,
   Valproate
2. Tricyclic agents

3. Capsaicin cream

Allodynia:

Plastic film (Opsite) on legs.

Restless Legs :

Clonazepam

Painful Cramps :

Quinine sulphate.

MANAGEMENT OF DIABETIC FOOT ULCERS

PREVENTION

Every diabetic patient should have an annual foot examination by a physician and if any risk factors are present, foot examination every 3 months.

Self-examination should be done daily.

Hyperglycemia should be controlled to slow down the development of Neuropathy.

Patients with signs of increased plantar pressure should wear protective footwear appropriately designed to cushion and redistribute weight.

Dryness should be treated with petroleum-based products.

Fungal infection should be treated with topical agents.

Toenail cutting should be done by patients only if there is no neuropathy.

For neuropathic patients regular chiropody is advised, with removal of callus if any.

TREATMENT OPTIONS

A. DRESSINGS

1. Alginates

Alginates are naturally occurring polysaccharides composed of the sugars mannuronic acid and guluronic acid.

They form a hydrophilic gel on the wound creating a moist environment.

They are best suited for treating moderately to highly exudative wounds.

2. Hydrocolloids

Hydrocolloids are based on the gelling agent sodium carboxy-methylcellulose combined with absorbent polymers and an adhesive.

They create a moist wound environment and retain growth factors under the dressing.

They may be used in Wagner stage I or II ulcers.

3. Hydrogels

Hydrogel dressings are composed of hydrophilic areas which absorb and retain significant volumes of water.

They rehydrate the wound and facilitate autolysis thereby promoting debridement

4. Films

They are semipermeable membranes, which form a bacterial barrier.

5. Foams

Foams absorb moderate amounts of fluid.

They are suited for exudating wounds.

Felt foams are applied to offload pressure points.

6. **Medicated dressings**

   They contain bactericidal agents, which are slowly released into the wound.

B. PHYSICAL THERAPIES

1. Hyperbaric oxygen
2. Lasers
3. Magnetic stimulation
4. Ultrasound stimulation
5. Vacuum devices
6. Warming

Hyperbaric oxygen therapy has been used to promote wound healing.

Vacuum-assisted dressings appear to be a promising way of treatment

Ultrasound and magnetic stimulation are not popular for treating diabetic foot ulcer.

C. BIOLOGICAL THERAPIES

1. Cytokines
2. Growth factors
3. Protease inhibitors
4. Epidermal allograft
5. Dermal allograft
6. Maggot therapy
7. Cadaver graft

Dermagraft consisting of neonatal fibroblasts in a bioabsorbable mesh is emerging as a new approach.

Vivoderm autograft, consisting of autologous cultured keratinocytes in a hyaluronic acid membrane represents another exciting and emerging innovation.

Platelet Derived Growth Factor and **Granulocyte colony stimulating factor** have also been used with good results.

Maggots have a voracious appetite for devitalized tissue while avoiding healthy tissue. Larvae are applied to the wound surface and covered with a hydrocolloid dressing. They can be removed when all the slough is cleared.

D. SURGICAL INTERVENTIONS

1. Debridement and drainage
2. Excision of necrotic tissue.
3. Skin grafting
4. Revascularisation
5. Amputations

LOCAL CARE OF THE DIABETIC FOOT

Grade 0 Foot:

A Grade 0 foot is the foot at risk without ulceration. The Grade 0 foot by definition has sensory neuropathy and is characterized by the presence of one or more risk factors. Areas of sensory neuropathy should be carefully mapped out so as to identify at risk areas of the foot. The foot should be closely inspected for corns or calluses.

Patients should be educated as to the risks associated with the neuropathic foot and the early signs of inflammation, irritation and infection. They should also be educated about the early treatment of these conditions.

Prevention is the hallmark of management of these feet and should include daily inspection by the patient or family member. High plantar foot pressures can be accommodated with shoegear modification, orthoses and padded hosiery.

An additional technique used to modify pressure points is the habit of changing ones' shoes every four hours. Changing shoes regularly also afford patients the opportunity to inspect their feet frequently.

Regular visits to the foot specialist should be a part of the patient's routine. The foot should be inspected at every visit. Pulses should be palpated. Any callus should be trimmed. Proper care of the nails and skin should be instructed to the patient. Moisturizing creams should be prescribed for dry, scaly skin so as to avoid fissures.

The interdigital spaces should also be inspected for cracking or presence of tinea pedis.

Grade 1 Foot:

Grade 1 ulcerations are those that have penetrated beyond the epidermis and are indicative of two or more risk factors: peripheral sensory neuropathy and at least one other risk factor. Ulcerations should be evaluated for size,

depth and location. The structures involved and the presence of infection should be carefully noted. The ulceration is usually colonized with multiple organisms representing primarily the skin flora.

Ulcers should be debrided to remove any hyperkeratotic tissue. Debridement allows for better visualization of the ulcer field and proper assessment of the depth of the defect. Debridement may be enzymatic, autolytic, mechanical and surgical.

Surgical debridement has been shown to be the best method for creating the proper environment for wound healing. Sharp debridement may be done with a scalpel and should be carried down through any necrotic or nonviable tissue until a bleeding ulcer bed is created. The debridement should also be carried to the wound margins, care being taken to uncover any undermining of the wound edges. It should be done in the operating theatre under suitable anesthesia.

The foot and specially the wound must be offloaded to eliminate all pressures from the site of ulceration. The gold standard for eliminating pressure is total non-weight-bearing, either with crutches or a walker. Most patients are unlikely to comply with this regimen entirely. Total contact casting, commercially available removable boots, half shoes and felted foam dressings have been described as ways of off-loading the foot and reducing the pressures.

The felted foam dressing is a simple approach towards offloading the insensate foot with a plantar ulcer. Its ease of use, cost effectiveness and reproducibility make it an important adjunct to treatment. ¼- inch felted foam, which is essentially a ¼ – inch piece of foam laminated to a thin layer of felt, rubber cement, a self-adhering gauze and tape are required.

Prior to application the wound needs to be measured. The appropriate aperture may be cut into the dressing, specifically the felted foam. Care must be taken to skive the edges of the dressing with a scissor. This is done on the edges of the dressing, as well as along the aperture closest to the actual ulcer. This step is performed so as to not to leave an "edge effect".

The foot is painted with rubber cement, along with both sides of each piece of the felted foam. Once this has dried, the two ¼ inch pieces of felted foam are laminated together, with the foam always on the bottom and the felt on the top. The felt top layer is attached directly to the skin. The self-adherent gauze is then wrapped around the foot to secure the dressing.

A window may be cut into the plantar surface of the dressing, allowing not only dressings, but also allowing direct visualization and assessment of the lesion. A surgical healing shoe is then dispensed to the patient, to be worn whenever ambulating. These pads are often left in place for approximately 10 to 14 days. Reapplication is done if necessary.

Advantages are easy daily dressings and frequent wound inspection. The disadvantage is that the patient has to keep the foot dry. Limited ambulation is absolutely necessary.

The ulceration is dressed daily to provide a moist environment conducive for wound healing. Infection is best treated with systemic appropriate antibiotics after testing for sensitivity.

Recurrent ulcerations may warrant surgical correction of the underlying deformity. Digital arthroplasties, metatarsal osteotomies, metatarsal head resections, midfoot and hindfoot ostectomies, joint arthrodeses and Achilles tendon lengthening have all been proved useful.

Grade 2 Foot:

Continued weight bearing on grade 1 lesions will cause ulcerations to become deeper. They will go beyond the dermis and can involve deeper structures. At this point the lesion is considered grade 2. Radiographs should be taken in all grade 2 ulcerations to evaluate for osteomyetlitis. To be considered a grade 2 lesion, films should be negative for osteomyelitis and bone cannot be probed. The ulcer base and periphery should be gently probed with a stainless steel blunt probe to determine undermining or the presence of any penetrating sinus tracts and involvement of deeper structures. This has 80%

specificity and 89% positive predictive value for diagnosing osteomyelitis. Radionuclide scans, MRIs, and bone biopsies are sometimes recommended for diagnosing osteomyelitis.

Hospitalization, complete bed rest, and empiric use of broad-spectrum antibiotics are often needed. Once the patient is hospitalized, the foot should be drained dependently and packed open. This is preferably done in the operating room. Any sinus tract should be explored and drained, and all nonviable tissue should be excised. This is done thoroughly and aggressively. Deeper cultures should be taken at this time. Preferably tissue should be sent instead of a swab. Intravenous antibiotics can then be given according to the sensitivity.

An aggressive incision and drainage or debridement of a foot with underlying ischemia should not be delayed. Adequate drainage of the infected foot is imperative. Consultation with a vascular surgeon as well as arterial studies should be initiated immediately. But surgery should not be delayed. Delaying surgery may lead to further tissue loss and potential limb loss, even with grade 2 lesions. Dressing changes and wound packing should be done at least twice a day. Subsequent bedside debridements while waiting to perform definitive closure are often necessary.

Once the tissues appear to be granulating, thought can be given to a definitive treatment of the ulceration and any underlying bony deformity. Some clinicians prefer to wait for secondary wound healing to take place. The myriad of topical agents available today may hasten the healing process. Healing requires prolonged periods of non-weight-bearing, and resultant area of scar tissue may not be suited for weight bearing. It is therefore advisable to close wounds primarily whenever possible.

Not all grade – 2 ulcerations require hospitalization. Same principles of treating grade 1 ulcerations, namely strict adherence to non-weight bearing and weekly debridement must be followed. Dressings should be changed more regularly, often twice a day. Oral antibiotics, although often not necessary in grade 1 ulcerations, are more commonly prescribed in grade 2 ulcerations.

Grade 3 Foot:

Deep infection that results in abscess formation and bone involvement is characteristic of the grade 3 foot. It may result from unresponsive grade 2 ulcerations, aggressive bacterial infections, or not uncommonly, puncture wounds resulting in direct inoculation of bone. Hospitalization and surgical intervention are required.

Adequate drainage of infection is absolutely essential in managing grade 3 lesions. The sinus tract must be explored and any undrained abscess or devitalized tissue must be thoroughly debrided. In cases of severe infection, open ray amputations become necessary to control the spread of infection.

Once the infection has cleared and healthy granulation tissue appears, thought can be given to surgical reconstruction of the wound and the foot. Delayed primary closure or more complicated reconstructive surgery like additional bone resections, tissue flaps, or skin grafts may be done. These lesions often require maximum use of all members of the diabetic foot team.

The long-term management of the grade 3 foot emphasizes prevention of transfer ulcerations. Because ablative surgery is common in the grade 3 foot, transfer of pressure to adjacent areas of the foot is expected. Prevention of chronically recurrent ulcerations requires the use of appropriate orthoses, prescription footwear and regular podiatry visits.

At each visit, orthoses and shoes should be inspected for signs of early wear and breakdown and replaced immediately when found to be worn down. Goal of long-term care is to distribute plantar foot pressures more evenly along the entire plantar aspect of the foot, thus avoiding concentration of pressure over any one focal area.

Grade 4 Foot

These are particularly challenging, Variety of underlying risk factors are present, making overall management difficult. Peripheral vascular disease, osteomyelitis, sepsis and extensive tissue loss necessitates the cooperation of all members of the limb salvage team. Consultations with vascular surgeons, podiatrists, plastic surgeons, and orthopedic surgeons are often required. The primary goal in the management of these lesions is to limit tissue loss.

Minor trauma in the face of severe arterial insufficiency can result in gangrene. It most commonly occurs at the distal end of extremities since this is where end-arteries are typically found. Lack of adequate perfusion and oxygenation will initially cause focal necrosis. This is most commonly referred to as dry gangrene.

Overwhelming infection may also result in gangrenous changes either from marked edema of local tissue or from infective vasculitis. Infective vasculitis will typically lead to wet gangrene.

Initial therapy depends on identifying the underlying cause. Gangrene resulting from arterial insufficiency should be treated with immediate vascular assessment and lower extremity revascularization when possible to minimize tissue loss.

Grade 5 Foot

Extensive necrosis of the foot characterizes the grade 5 foot. Direct cause is arterial occlusion and failure of arterial inflow. Primary amputation is the only treatment for extensive gangrene. These patients should undergo vascular assessment and revascularization whenever possible to reduce tissue loss and to allow for amputation at the most distal level that will support healing.

Diabetic foot ulcerations may be neuropathic, ischemic or neuroischemic.

NEUROPATHIC ULCERS

Even with adequate arterial inflow, neuropathic ulcers may remain unchanged for months secondary to impaired inflammatory and healing responses.

Infection requires prompt management when it arises. Aggressive debridement with removal of excessive callus and all necrotic debris is essential.

Protecting the weight bearing surface of the foot and offloading the mechanical stress is of paramount importance.

Bed rest for 6-12 weeks is an ideal but poorly tolerated method to promote healing.

Total contact casting is an alternative which uses a protective short leg cast to mechanically offload the foot at the ulcer site. Scotch cast boots and healing sandals also relieve pressure and allow easy access for dressings.

Provided there is adequate vascular supply surgical correction of architectural deformities can be performed. Correction of clawed toes,

arthodesis of interphalangeal joints or resection of the prominent bone can be done.

ISCHEMIC ULCERS AND GANGRENE :

If the ulcer is small and superficial, a trial of conservative treatment under close supervision can be done. If there is no rapid healing, angiography is carried out and vascular reconstruction is done when appropriate. This may involve angioplasty, stenting, bypass surgery or a combination.

Bypass from the femoral or popliteal arteries to tibial, peroneal or pedal vessels are often required. Autogenous vein is used for the bypass surgery with 3-5 year patency rate of 80-90%.

In the presence of dry gangrene, evaluation for revascularisation should be undertaken with amputations delayed until this can be safely performed.

INVASIVE FOOT INFECTION :

Infection in diabetic foot may progress rapidly to acute foot sepsis or systemic sepsis. Traditional signs of infection may be absent or attenuated. Hyperglycemia or even ketoacidosis may occur.

The initial management is resuscitation and control of the foot infection. Drainage and debridement of necrotic tissues are the primary goals. Wounds should be opened to permit free drainage. Involved bones or joints should be resected.

Partial foot amputations may be warranted to control sepsis. These amputations are left open. Refashioning of the stump and

definitive amputation follows when the infection has been controlled and the metabolic derangements improve.

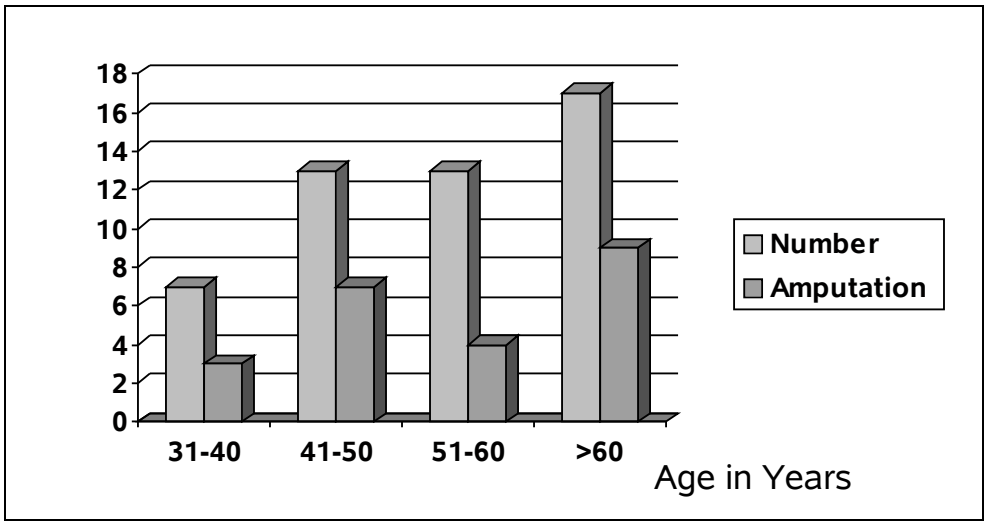
AMPUTATIONS

Amputations are indicated for removal of gangrenous or necrotic tissue. With normal circulation, amputation can usually be performed just proximal to the extent of the necrotic tissue. In vascular compromise, the amputation should be performed at the level that balances the highest chance of healing with the greatest function.

A transmetatarsal amputation is considered if multiple toes or the distal forefoot is involved. If ischemic disease or infection involves the proximal foot, a below-knee amputation is indicated in the ambulatory patient.

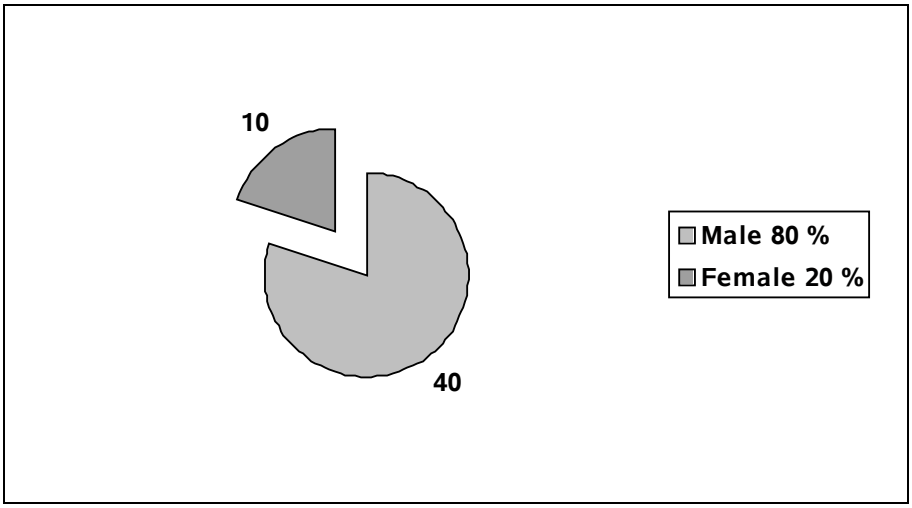
Above-knee amputation may be appropriate in chronically non-ambulatory patients, patients with fixed knee contractures or patients with marginal and unreconstructable distal circulation.

DIABETIC ULCER - AGE WISE

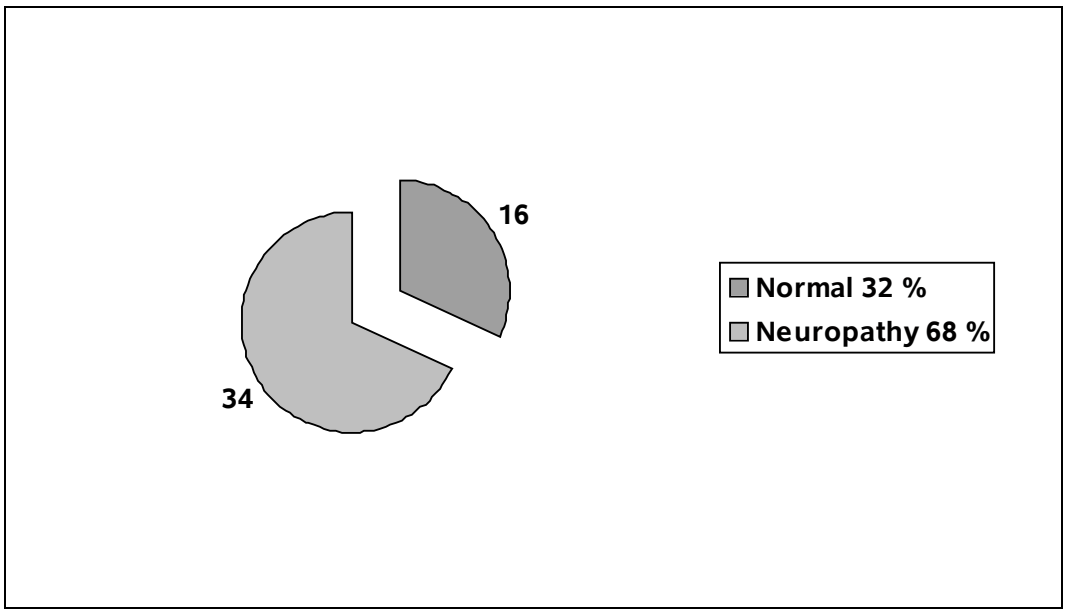


No	Age Group in Years	No.of patients	No. of Amputations	Percentage
1.	31-40	7	3	42.9 %
2.	41- 50	13	7	53.8 %
3.	51-60	13	4	30.8 %
4.	>61	17	9	52.9 %
Total		50	23	45.08 %

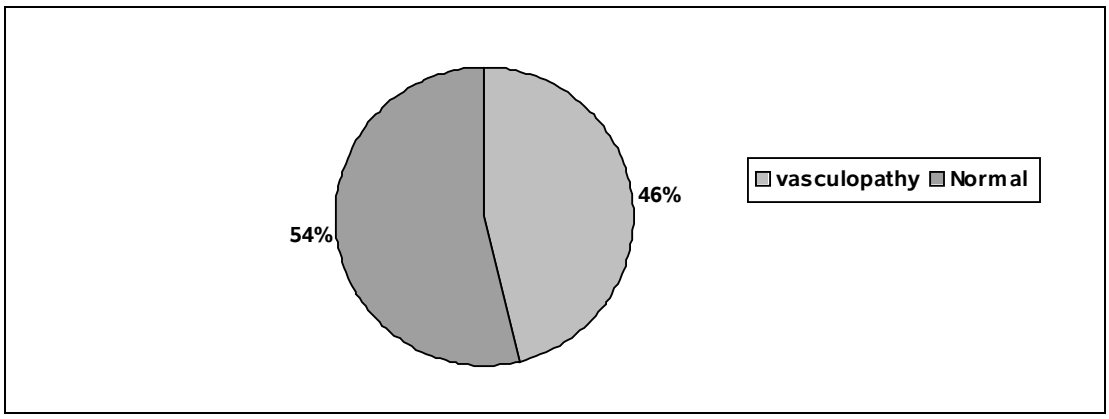
DIABETIC ULCER - Sex wise incidence



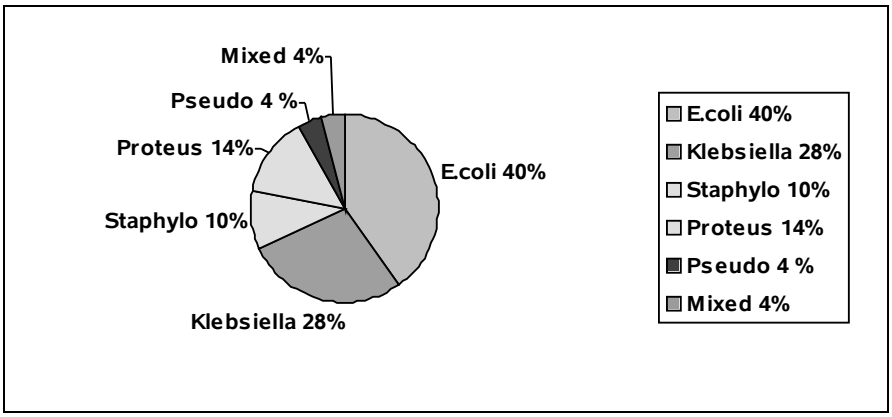
Neuropathy in diabetics



Macrovascular Disease in Diabetics



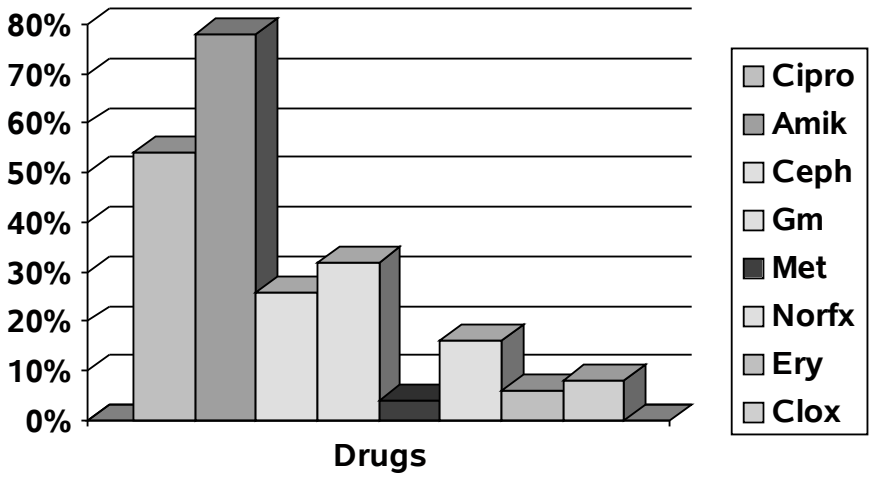
INFECTIVE ORGANISM IN DIABETIC ULCER



INFECTIVE ORGANISM

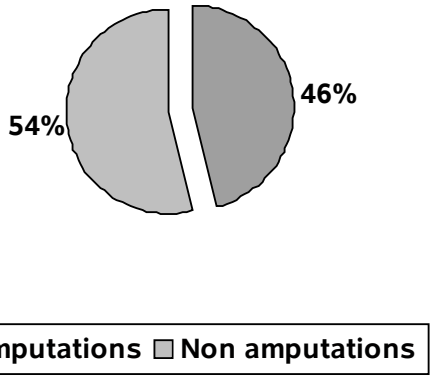
S.No	Organism	No of Patients	Percentage
1	E. coli	20	40 %
2	Klebsiella	14	28 %
3	Staphylococcus	5	10 %
4	Proteus	7	14 %
5	Pseudomonas	2	4 %
6	Mixed	2	4 %
	Total	50	100 %

SENSITIVE DRUG IN DIABETIC ULCER



S. No	Sensitive Drug	No. of patients	Percentage
1	Ciprofloxacin	27	54
2	Amikacin	39	78
3	Cephalexin	13	26
4	Gentamycin	16	32
5	Metronidazole	02	4
6	Norfloxacin	08	16
7	Erythromycin	03	6
8	Cloxacillin	04	8

MODE OF TREATMENT



Total No. of Patients	Amputations		Non amputations	
	Number	Percentage	Number	Percentage
50	23	46%	27	54%

S. No.	Level of Amputation	No. of Patients	Percentage
1	Above Knee	1	4.3 %
2	Through Knee	1	4.3 %
3	Below Knee	11	47.8 %
4	Toe Disarticulation	10	43.5 %

CONCLUSION

The epidemiology of this study revealed that the patients with diabetic foot ulcers constitute 0.2% of inpatients per year.

Diabetic foot ulcer is the commonest cause of foot ulcers in Thanjavur Medical College Hospital, Thanjavur. In this study, the incidence of Diabetic foot ulcers increased with age, which correlates with the literature.

In this study, the predominant affected sex were males, with male:female ratio of 4:1 which contradicts the western study where the affected gender is commonly female with male : female ratio of 1 : 2.5

Pus culture and sensitivity done for diabetic foot ulcers reveal E.coli as the commonest organism (40%) when compared to standard study which mention mixed infection – staphyloccus and proteus as the commonest organisms.

Skin biopsy done in these patients reveal neuropathic changes in 34 patients. This result is comparable with the standard study.

46% of patients had vaso-occlusive disease which is slightly higher compared to the western study which states the prevalence of vaso-occlusive disease as 30%. But this series is too small to comment.

3 out of 50 patients showed osteomyelitic changes emphasizing that diabetic ulcer patients are prone for osteomyelitis of the underlying bone.

46% of patients with diabetic foot ulcer needed either minor or major amputation, which correlates with the standard study.

There is an increasing incidence of amputations in the younger age group of 41-50 revealing that diabetics who smoke are more prone for ischemia and gangrene at an earlier age.

This study confirms that prevention is the most effective way of dealing with the diabetic foot ulcer and that early recognition and liberal debridement with proper antibiotic cover and excellent diabetic control will reduce the incidence of amputations. But amputations have a major role in the treatment of diabetic foot ulcers since patients report late with life threatening complications and uncontrolled diabetes.

A specialist chiropodist is an integral part of the diabetes team to ensure regular and effective chiropody for prevention of diabetic foot ulcer.

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PROFORMA

NAME **AGE/SEX** **I.P.NO**

UNIT/WARD **OCCUPATION**

HISTORY:

LEG ULCER DURATION

SITE

PIGMENTATION

ITCHING

BLEEDING

DISCHARGE

CLAUDICATION

SENSORY LOSS

DEFORMITY

PAST HISTORY:

DIABETES **DURATION**

TREATMENT -yes/no

HYPERTENSION

ISCHEMIC HEART DISEASE

TUBERCULOSIS

PERSONAL HISTORY:

SMOKING

ALCOHOL

EXAMINATION

GENERAL:

ANEMIA

FEVER

JAUNDICE

LOCAL:

INSPECTION	**PALPATION**
SITE	**TEMPERATURE**
SIZE	**TENDERNESS**
SHAPE	**EDGE**
MARGIN	**BASE**
FLOOR	**SURROUNDING AREA**
SURROUNDING AREA	**REGIONAL NODE**

SPECIAL TEST:

SENSORY TESTING	**MOTOR TESTING**
LIGHT TOUCH	**POWER**
CRUDE TOUCH	**DEFORMITY**
JOINT SENSATION	
VIBRATION SENSATION	

INVESTIGATION:

URINE: ALBUMIN

SUGAR

DEPOSITS

BLOOD: Hb% TC DC

ESR PLATELET

BLOOD: SUGAR UREA

SERUM: CREATININE

SODIUM

POTASSIUM

ECG & CHEST XRAY:

PUS CULTURE AND SENSITIVITY:

ORGANISM:

SENSITIVE DRUG: Cip, A, Ce, Gm, Nrfx, Er, Clox, Met

SKIN BIOPSY:

NEUROPATHIC CHANGES:- Present / Absent

XRAY OF LOCAL PART:

OSTEOMYELITIS:- Present / Absent

SUBCUTANEOUS GAS: Present / Absent

BONY DEFORMITIES : Present / Absent

COLOUR DOPPLER STUDY:

FINAL DIAGNOSIS:

TREATMENT:

Type: CONSERVATIVE DEBRIDEMENT /

AMPUTATION

LEVEL: TOE / SYMES / BK / AK

HOSPITAL STAY: